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*DB=USPT,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ*L8 (lung or pulmonary) same (cancer or tumor or tumour) and (alphaV or 'alpha v')47 L8L7 L6 and (alphaV or 'alpha v')1 L7L6 L5 and (pulmonary or lung) same (fibrosis or fibrotic)62 L6L5 rgd and (fibrosis or fibrotic)174 L5*DB=USPT; PLUR=YES; OP=ADJ*L4 (alphav or 'alpha v') and (fibrosis or fibrotic)16 L4*DB=USPT,EPAB,DWPI; PLUR=YES; OP=ADJ*L3 (alphav or 'alpha v') same (lung or pulmonary)10 L3L2 (alphav or 'alpha v') same (antibod\$) and (treat\$ or therap\$ or suppress\$ or inhibit\$ or prevent\$) same (fibrosis or fibrotic)2 L2L1 (alphav or 'alpha v') same (fibrosis or fibrotic)14 L1

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☐ 1. Document ID: KR 2002015704 A DE 19929410 A1 WO 200100660 A1 AU 200062630 A NO 200106341 A EP 1189930 A1 CZ 200104484 A3 BR 200011954 A SK 200101872 A3

L2: Entry 1 of 2

File: DWPI

Feb 28, 2002

DERWENT-ACC-NO: 2001-113366

DERWENT-WEEK: 200258

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TITLE: New octapeptide compounds as alpha v beta 6 integrin inhibitors useful for treating and diagnosing heart disease, tumors, osteoporosis, fibrosis, inflammation, infection and psoriasis

INVENTOR: DIEFENBACH, B; GROTH, U ; JONCZYK, A ; ZISCHINSKY, G

PRIORITY-DATA: 1999DE-1029410 (June 26, 1999)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
KR 2002015704 A	February 28, 2002		000	C07K007/06
DE 19929410 A1	December 28, 2000		033	C07K007/06
WO 200100660 A1	January 4, 2001	G	000	C07K007/06
AU 200062630 A	January 31, 2001		000	C07K007/06
NO 200106341 A	February 25, 2002		000	C07K000/00
EP 1189930 A1	March 27, 2002	G	000	C07K007/06
CZ 200104484 A3	April 17, 2002		000	A61K038/04
BR 200011954 A	May 7, 2002		000	C07K007/06
SK 200101872 A3	May 9, 2002		000	C07K007/06

INT-CL (IPC): A61 K 38/04; A61 K 38/08; A61 P 7/02; C07 K 0/00; C07 K 7/06

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KWIC	Draw Desc	Image
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☒ 2. Document ID: NZ 502546 A WO 9907405 A1 AU 9887743 A EP 996460 A1 BR 9814040 A CN 1267224 A CZ 200000413 A3 HU 200003547 A2 MX 2000001196 A1 KR 2001022740 A JP 2001513333 W AU 739283 B US 6316601 B1 US 2001056076 A1 US 2002004482 A1

L2: Entry 2 of 2

File: DWPI

Feb 1, 2002

DERWENT-ACC-NO: 1999-167213

DERWENT-WEEK: 200214

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TITLE: Preventing and treating acute lung injury and pulmonary fibrosis - with antagonists of integrin (v6)

INVENTOR: DEAN, S; ROBERT, P ; XIAOZHU, H ; HUANG, X ; PYTELA, R ; SHEPPARD, D

PRIORITY-DATA: 1997US-055060P (August 8, 1997), 1998US-0130870 (August 7, 1998),

1999US-0365695 (August 2, 1999), 2001US-0818416 (March 27, 2001)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
NZ 502546 A	February 1, 2002		000	A61K039/395
WO 9907405 A1	February 18, 1999	E	023	A61K038/16
AU 9887743 A	March 1, 1999		000	A61K038/16
EP 996460 A1	May 3, 2000	E	000	A61K038/16
BR 9814040 A	October 3, 2000		000	A61K038/16
CN 1267224 A	September 20, 2000		000	A61K038/16
CZ 200000413 A3	November 15, 2000		000	A61K038/16
HU 200003547 A2	February 28, 2001		000	A61K038/16
MX 2000001196 A1	October 1, 2000		000	A61K038/16
KR 2001022740 A	March 26, 2001		000	A61K038/16
JP 2001513333 W	September 4, 2001		026	C12N015/09
AU 739283 B	October 11, 2001		000	A61K038/16
US 6316601 B1	November 13, 2001		000	C07K016/28
US 2001056076 A1	December 27, 2001		000	A61K048/00
US 2002004482 A1	January 10, 2002		000	A61K038/16

INT-CL (IPC): A61 K 31/70; A61 K 31/711; A61 K 38/00; A61 K 38/16; A61 K 38/17; A61 K 39/395; A61 K 45/00; A61 K 48/00; A61 P 11/00; C07 H 21/04; C07 K 14/00; C07 K 14/435; C07 K 14/47; C07 K 16/18; C07 K 16/28; C07 K 17/00; C12 N 5/10; C12 N 5/12; C12 N 15/09; C12 P 21/08; C12 P 21/08; C12 R 1:91

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments
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FIBROSES.DWPI,EPAB,USPT.	169
FIBROTIC.DWPI,EPAB,USPT.	2315
FIBROTICS.DWPI,EPAB,USPT.	5
ANTIBOD\$	0
ANTIBOD.DWPI,EPAB,USPT.	352
ANTIBODANTIBODA.DWPI,EPAB,USPT.	1
((ALPHAV OR 'ALPHA V') SAME (ANTIBOD\$) AND (TREAT\$ OR THERAP\$ OR SUPPRESS\$ OR INHIBIT\$ OR PREVENT\$) SAME (FIBROSIS OR FIBROTIC)).USPT,EPAB,DWPI.	2

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L3: Entry 10 of 10

File: DWPI

May 26, 1998

DERWENT-ACC-NO: 1996-171609

DERWENT-WEEK: 199831

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TITLE: Recombinant adenovirus vectors for gene therapy - used in the treatment of glioma, melanoma and cystic fibrosis

Basic Abstract Text (2):

USE - The recombinant viruses and vectors may be used in gene therapy (claimed). For example, a recombinant adenovirus having a penton base molecule recognised by alpha v beta 3 receptors can be used to treat melanoma or glioma, and a recombinant adenovirus recognised by alpha 3 beta 1 receptors and expressing the cystic fibrosis transmembrane regulator (CFTR) gene can be used to treat cystic fibrosis by delivery to the epithelial cells of the lungs. The recombinant adenoviruses may also be used to treat blood related diseases, pathogenic infections including HIV infection, and angiogenesis.

Equivalent Abstract Text (3):

USE - The recombinant viruses and vectors may be used in gene therapy (claimed). For example, a recombinant adenovirus having a penton base molecule recognised by alpha v beta 3 receptors can be used to treat melanoma or glioma, and a recombinant adenovirus recognised by alpha 3 beta 1 receptors and expressing the cystic fibrosis transmembrane regulator (CFTR) gene can be used to treat cystic fibrosis by delivery to the epithelial cells of the lungs. The recombinant adenoviruses may also be used to treat blood related diseases, pathogenic infections including HIV infection, and angiogenesis.

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L6: Entry 2 of 62

File: USPT

Sep 10, 2002

DOCUMENT-IDENTIFIER: US 6447753 B2

TITLE: Porous particles for deep lung delivery

Brief Summary Text (5):

Inhaled aerosols have been used for the treatment of local lung disorders including asthma and cystic fibrosis (Anderson, et al., Am. Rev. Rev. Respir. Dis., 140:1317-1324 (1989)) and have potential for the systemic delivery of peptides and proteins as well (Patton and Platz, Advanced Drug Delivery Reviews, 8:179-196 (1992)). However, pulmonary drug delivery strategies present many difficulties for the delivery of macromolecules; these include protein denaturation during aerosolization, excessive loss of inhaled drug in the oropharyngeal cavity (typically exceeding 80%), poor control over the site of deposition, irreproducibility of therapeutic results owing to variations in breathing patterns, the quick absorption of drug potentially resulting in local toxic effects, and phagocytosis by lung macrophages.

Detailed Description Text (42):

The porous particles may include a therapeutic agent for local delivery within the lung, such as agents for the treatment of asthma, emphysema, or cystic fibrosis, or for systemic treatment. For example, genes for the treatment of diseases such as cystic fibrosis can be administered.

Other Reference Publication (30):

Anderson, P.J., et al., "Effect of Cystic Fibrosis on Inhaled Aerosol Boluses," Am. Rev. Respir. Dis., 140:1317-1324 (1989).

Other Reference Publication (42):

Barrera, D.A., et al., "Synthesis and RGD Peptide Modification of a New Biodegradable Copolymer: Poly(lactic acid-co-lysine)," J. Am. Chem. Soc., 115:11010-11011 (1993).

CLAIMS:

9. The composition of claim 4 wherein the agent is an agent for the treatment of asthma, emphysema, or cystic fibrosis.
17. The composition of claim 12 wherein the agent is an agent for the treatment of asthma, emphysema, or cystic fibrosis.
39. The method of claim 34 wherein the agent is an agent for the treatment of asthma, emphysema, or cystic fibrosis.
49. The method of claim 44 wherein the agent is an agent for the treatment of asthma, emphysema, or cystic fibrosis.

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L6: Entry 23 of 62

File: USPT

Aug 21, 2001

DOCUMENT-IDENTIFIER: US 6277812 B1

TITLE: Methods for inhibiting TGF-.beta. activity

Detailed Description Text (2):

Increased TGF-.beta. production has been found to be an important element in a number of fibrotic diseases that are characterized by an accumulation of extracellular matrix components (Border and Ruoslahti, 1992). Besides fibronectin, collagens, and tenascin (Ignotz and Massague, 1986; Varga et al., 1987; Pearson et al., 1988), TGF-.beta. also upregulates the expression of proteoglycans (Bassols and Massague, 1988). In mesangial cells both decorin and biglycan can increase as much as 50-fold after induction by TGF-.beta. (Border et al., 1990a), whereas in fibroblasts only biglycan seems to be elevated (Romaris et al., 1992; Kahari et al., 1991). Fibromodulin has not been studied in this regard. TGF-.beta. plays a pivotal role in the pathogenesis of experimentally induced glomerulonephritis, the most critical manifestation of which is the accumulation of extracellular matrix in the glomeruli (Border et al., 1990). A recent study shows that injection of recombinant decorin into glomerulonephritic rats can suppress the matrix accumulation (Border et al., 1992). The present invention indicates that fibromodulin can be even more effective in that situation. The TGF-.beta. neutralizing activities of the decorin-type proteoglycans indicates that new types of therapeutics can be developed based on these molecules.

Detailed Description Text (13):

The invention additionally provides a method of treating a pathology caused by a TGF-.beta.-regulated activity comprising contacting the TGF-.beta. with a purified polypeptide, wherein the polypeptide comprises the TGF-.beta. binding domain of a protein and wherein the protein is characterized by a leucine-rich repeat of about 24 amino acids, whereby the pathology-causing activity is prevented or reduced. While the method is generally applicable, specific examples of pathologies which can be treated include cancer, a fibrotic disease, and glomerulonephritis. In fibrotic cancer, for example, decorin can be used to bind TGF-.beta., destroying TGF-.beta.'s growth stimulating activity on the cancer cell. Other proliferative pathologies include rheumatoid arthritis, arteriosclerosis, adult respiratory distress syndrome, cirrhosis of the liver, fibrosis of the lungs, post-myocardial infarction, cardiac fibrosis, post-angioplasty restenosis, renal interstitial fibrosis and certain dermal fibrotic conditions such as keloids and scarring.

Detailed Description Text (19):

In addition, the present invention further relates to a pharmaceutical composition containing decorin or its functional equivalent, such as biglycan or fibromodulin, and a pharmaceutically acceptable carrier useful in the above methods. Pharmaceutically acceptable carriers include, for example, hyaluronic acid, and aqueous solutions such as bicarbonate buffers, phosphate buffers, Ringer's solution and physiological saline supplemented with 5% dextrose or human serum albumin, if desired. The pharmaceutical compositions can also include other agents that promote wound healing known to those skilled in the art. Such agents can include, for example, biologically active chemicals and polypeptides, including RGD-containing polypeptides attached to a biodegradable polymer as described in PCT WO 90/06767 published on Jun. 28, 1990, and incorporated herein by reference. Such polypeptides can be attached to polymers by any means known in the art, including covalent or ionic binding, for example.

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L6: Entry 59 of 62

File: USPT

Aug 5, 1997

DOCUMENT-IDENTIFIER: US 5654270 A

TITLE: Use of fibromodulin to prevent or reduce dermal scarring

Detailed Description Text (2):

Increased TGF- β production has been found to be an important element in a number of fibrotic diseases that are characterized by an accumulation of extracellular matrix components (Border and Ruoslahti, 1992). Besides fibronectin, collagens, and tenascin (Igotz and Massague, 1986; Varga et al., 1987; Pearson et al., 1988), TGF- β also upregulates the expression of proteoglycans (Bassols and Massague, 1988). In mesangial cells both decorin and biglycan can increase as much as 50-fold after induction by TGF- β . (Border et al., 1990a), whereas in fibroblasts only biglycan seems to be elevated (Romaris et al., 1992; Kahari et al., 1991). Fibromodulin has not been studied in this regard. TGF- β plays a pivotal role in the pathogenesis of experimentally induced glomerulonephritis, the most critical manifestation of which is the accumulation of extracellular matrix in the glomeruli (Border et al., 1990). A recent study shows that injection of recombinant decorin into glomerulonephritic rats can suppress the matrix accumulation (Border et al., 1992). The present invention indicates that fibromodulin can be even more effective in that situation. The TGF- β neutralizing activities of the decorin-type proteoglycans indicates that new types of therapeutics can be developed based on these molecules.

Detailed Description Text (13):

The invention additionally provides a method of treating a pathology caused by a TGF- β -regulated activity comprising contacting the TGF- β with a purified polypeptide, wherein the polypeptide comprises the TGF- β binding domain of a protein and wherein the protein is characterized by a leucine-rich repeat of about 24 amino acids, whereby the pathology-causing activity is prevented or reduced. While the method is generally applicable, specific examples of pathologies which can be treated include cancer, a fibrotic disease, and glomerulonephritis. In fibrotic cancer, for example, decorin can be used to bind TGF- β , destroying TGF- β 's growth stimulating activity on the cancer cell. Other proliferative pathologies include rheumatoid arthritis, arteriosclerosis, adult respiratory distress syndrome, cirrhosis of the liver, fibrosis of the lungs, post-myocardial infarction, cardiac fibrosis, post-angioplasty restenosis, renal interstitial fibrosis and certain dermal fibrotic conditions such as keloids and scarring.

Detailed Description Text (19):

In addition, the present invention further relates to a pharmaceutical composition containing decorin or its functional equivalent, such as biglycan or fibromodulin, and a pharmaceutically acceptable carrier useful in the above methods. Pharmaceutically acceptable carriers include, for example, hyaluronic acid, and aqueous solutions such as bicarbonate buffers, phosphate buffers, Ringer's solution and physiological saline supplemented with 5% dextrose or human serum albumin, if desired. The pharmaceutical compositions can also include other agents that promote wound healing known to those skilled in the art. Such agents can include, for example, biologically active chemicals and polypeptides, including RGD-containing polypeptides attached to a biodegradable polymer as described in PCT WO 90/06767, published on Jun. 28, 1990, and incorporated herein by reference. Such polypeptides can be attached to polymers by any means known in the art, including covalent or ionic binding, for example.

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L6: Entry 62 of 62

File: DWPI

Aug 14, 2002

DERWENT-ACC-NO: 1993-182240

DERWENT-WEEK: 200261

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TITLE: Prevention or reduction of dermal scarring - by administration of decorin or its functional equivalents bi-glycan or fibromodulin

Basic Abstract Text (2):

Also claimed are: a pharmaceutical compsn. comprising decorin or its functional equivalents and a carrier, e.g. hyaluronic acid; the compsn. may further comprise an RGD-contg. polypeptide attached to a biodegradable polymer; a method of treating a pathology caused by a transforming growth factor (TGF)-beta regulated activity comprising contacting the TGF-beta with a purified polypeptide comprising a TGF-beta binding domain of a protein characterised by a leucine-rich repeat of about 24 amino acids, whereby the pathology-causing activity is prevented or reduced, the protein may be e.g. decorin, biglycan or fibromodulin.

Basic Abstract Text (3):

USE/ADVANTAGE - Decorin binds and neutralises a variety of biological functions of TGF-beta, including the induction of extracellular matrix. Thus decorin can be used to prevent or reduce dermal scarring resulting from burns, skin injuries or surgery. The polypeptides can be used for treating TGF-beta pathologies such as rheumatoid arthritis, glomerulonephritic, arteriosclerosis, adult respiratory distress syndrome, cirrhosis of the liver, fibrotic cancer, fibrosis of the lungs, post-myocardial infarction, cardiac fibrosis, post-angioplasty restenosis, renal interstitial fibrosis or dermal fibrotic conditions (claimed)

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PULMONARIES	0
PULMONARYS	0
CANCER.DWPI,EPAB,JPAB,USPT.	93005
CANCERS.DWPI,EPAB,JPAB,USPT.	21667
TUMOR.DWPI,EPAB,JPAB,USPT.	53934
TUMORS.DWPI,EPAB,JPAB,USPT.	33289
TUMOUR.DWPI,EPAB,JPAB,USPT.	22901
TUMOURS.DWPI,EPAB,JPAB,USPT.	12841
((LUNG OR PULMONARY) SAME (CANCER OR TUMOR OR TUMOUR) AND (ALPHA V OR 'ALPHA V')).USPT,JPAB,EPAB,DWPI.	47

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L8: Entry 23 of 47

File: USPT

Nov 16, 1999

DOCUMENT-IDENTIFIER: US 5985278 A

TITLE: Anti-.alpha.V-integrin monoclonal antibody

Brief Summary Text (7):

Studies in vivo also implicate .alpha.V.beta.3 in melanoma development. In the murine B16-F10 melanoma system, experimental lung metastasis could be suppressed by high levels of RGD-peptides (Hardan et al., 1993; Humphries et al., 1986), potent blockers of .alpha.v-integrin function. More recently, Felding-Habermann and colleagues have shown that .alpha.V-series integrins promote subcutaneous tumor growth of M21 human melanoma in immune-deficient mice. The M21 system is elegant, and consists of a suite of cells expressing different .alpha.V-series integrins (Kieffer et al., 1991; Felding-Habermann et al., 1992; Cheresch and Spiro, 1987). The parent, M21, expresses .alpha.V.beta.3 and .alpha.V.beta.5 (Wayner et al., 1991): it attaches to vitronectin and grows as a subcutaneous tumor. M21-L, a somatic variant of M21, has no detectable .alpha.V (Cheresch and Spiro, 1987): it cannot bind vitronectin and develops slow-growing tumors. M21-L4 is a transfectant of M21-L, stably re-expressing a full length .alpha.V-chain: it binds vitronectin and grows rapidly as a subcutaneous tumor (Felding-Habermann et al., 1992). Thus the presence of cell surface .alpha.V-integrins is directly correlated with M21 subcutaneous growth.

Drawing Description Text (3):

FIG. 1: Fluorescence Activated Cell Sorter (FACS) Analysis of the Alpha-V Group Antibodies and Controls to M21 and M21L Human Melanoma Cells

Drawing Description Text (4):

Cells were incubated with 10 g ml.sup.-1 primary antibodies, stained with fluorescently labelled secondary antibodies, counter stained with propidium iodide to allow gating of necrotic cells, and 10,000 cells per sample were analyzed. The open peak represents intensity of the second layer antibody alone. The closed peak, the intensity of the specifying primary and secondary together. Vertical axis shows cells per channel, horizontal axis shows log fluorescent intensity in that channel. M21 carries surface .alpha.v integrin, M21-L has none. The pattern of staining for the alpha-V group antibodies closely matches the LM142 (.alpha.v-specific) and LM609 (.alpha.v.beta.3-specific) stainings. Especially, they react with M21 but minimally with M21-L. Antibody 9.2.27 reacts with a surface proteoglycan. 14E2 and 21H6 recognize an otherwise undefined 200 kDa melanoma surface protein. Their staining patterns are discrete from those of the alpha-V group. Especially, they react similarly with both M21 and M21-L.

Drawing Description Text (5):

FIG. 2: The Alpha-V Group Antibodies Immunoprecipitate Similar Proteins

Drawing Description Text (19):

0.32.times.106 (.diamond-solid.,.box-solid.) or 1.times.10.sup.6 (.diamond.,.quadrature.) of M21 (.diamond-solid.,.diamond.) or M21L (.box-solid.,.quadrature.) cells were injected into the tail vein of nude mice. At the time shown, groups of animals were killed and the lungs examined for tumor nodules. All the M21 mice were killed at 42 days. For the M21-L mice, 3-6 mice were sacrificed at each point. The tumor burden after 6 weeks in the M21 mice was too high to count (>>250 per lung), so T-statistics are shown for the hypothesis that an M21-L group is from the same population as the control M21 group at 42 days at the <0.001 level (**) or <0.02 (*). Where both hi and lo injected groups have the same

significance only one is shown. The bars show the mean tumor number for the groups. Vertical axis: metastases/lung; horizontal axis: days post injection.

Detailed Description Text (3):

The alpha-V Group Monoclonal Antibodies React with Integrin .alpha.v-chain

Detailed Description Text (4):

Antibody screening by ELISA on purified .alpha.v.beta.3 and .alpha.IIb.beta.3 revealed five clones, 17E6, 20A9, 23G5, 14D9.F8 and 10G2 which reacted specifically with .alpha.v.beta.3 (Table 1). These MABs are termed "the alpha-V group". All were IgG1 isotype. In the same ELISA assay, anti-integrin antibodies of known specificity against the .alpha.v.beta.3 complex (LM609), the .alpha.v chains (LM142), the .alpha.v.beta.5 complex (P5H9), the .alpha.IIb.beta.3 complex (CP8), the .beta.3 chains (AP3) and the .beta.1 chains (P4C10), reacted as predicted from the literature (Table 1). In ELISA on fixed cells (CELISA), with cells expressing .alpha.v.beta.3 and .alpha.v.beta.5 (M21), .alpha.v.beta.5 but no .alpha.v.beta.3 (UCLAP3), neither .alpha.v.beta.3 nor .alpha.v.beta.5 (M21-L), and .alpha.IIb.beta.3 (M21-L-IIb), the .alpha.V group showed a reaction pattern consistent with their recognition of the .alpha.v-integrin chain and clearly distinct from a reaction with .beta.3, .beta.5, .beta.1, or other .alpha.-chains (Table 1).

Detailed Description Text (5):

The results corroborated the ELISA data with purified receptors. MABs with specificities for .beta.3, and GpIIb were also obtained in the screen (data not shown), and these reacted in a way clearly discrete from the alpha-v group. 17E6, 14D9.F8, 20A9 and 23G5 bound .alpha.v.beta.3 with similar apparent affinity. 50% binding was achieved at .about.10-20 ng ml.sup.-1 (.about.50-100 pM--similar to LM609). 10G2 bound similar to LM142 with about 10 times lower affinity). CP8, against .alpha.IIb.beta.3 and 14E2 (see below), showed minimal binding to .alpha.v.beta.3 at concentrations up to 100 nM.

Detailed Description Text (6):

The ability of the alpha-V group to recognize native .alpha.v-integrins was tested by FACS (FIG. 1; Table 2) and by immunoprecipitation from surface labelled cells (FIG. 2). In FACS analysis (FIG. 1), the .alpha.v-expressing line (M21) reacted strongly with 17E6, 14D9.F8, 20A9, 23G5, and with the .alpha.v-defining antibodies LM142 and LM609, moderately with 10G2, and also with the control MABs 14E2 and 21H6 and Mab 9.2.27. By contrast, .alpha.v-deficient variant (M21-L) reacted weakly with the alpha-V group and with LM142 and LM609, but showed similar reactivity as M21 with 14E2, 21H6 and 9.2.27. M21-L has an intracellular pool of .beta.3 subunits which were detected in FACS only when the cells were permeabilized (Table 2).

Detailed Description Text (7):

In FACS analysis of M21-L4 (.alpha.v-retransfected M21-L cells (Felding-Habermann et al., 1992)), the alpha-V group gave reaction patterns as on M21. The control vector transfectants, M21-L12 and the GpIIb transfectants, M21-L-IIb (Kieffer et al., 1991), showed no reactions with the alpha-V group (Table 1). UCLAP-3 adenocarcinoma reacted with the alpha-V group, with LM142 and P5H9, but not with LM609. UCLAP-3 does not express .beta.3 (see Background). The melanoma WM793 had the same reaction pattern as M21. In immunoprecipitation screening of M21 cells, the alpha-V group gave the same immunoprecipitation patterns as LM142 (anti-.alpha.v), and LM609 (anti .alpha.v.beta.3) (FIG. 2a). A strong broad band was seen at .about.92 kDa and a weaker band at .about.145 kDa, with weak accompanying bands at .about.100 kDa, a pattern characteristic of surface labelled .alpha.v.beta.3 and .alpha.v.beta.5 integrins (Wayner et al., 1991). When compared to the precipitation patterns on M21-L, none of the alpha-V group precipitated (data from 17E6 and 20A9 are shown), and neither did LM142 or LM609. (FIG. 2c). .beta.1-specific antibodies gave similar precipitation patterns from both cell lines. In M21-L, precipitation with anti-.beta.3 antibodies gave a band at .about.92 kDa, due to intracellular .beta.3-labelled in permeable (possibly necrotic) cells. UCLAP3 (FIG. 2d) gave no precipitate with LM609, but a .about.95 kDa/145 kDa complex was precipitated, by the alpha-V group and by LM142 (FIG. 2d). In summary, ELISA, CELISA, FACS analyses and immunoprecipitations of gave consistent reaction patterns and strongly suggested that MABs of the alpha-V group react with extracellular domains on human .alpha.v-integrin chains.

Detailed Description Text (10):

.alpha.v-integrins can function as receptors for vitronectin, so the alpha-V group was screened for their possible effects on cell attachment to vitronectin substrates. After integrin analysis by FACS, cells were tested in attachment assays (Table 2, FIG. 3). In FACS, human melanoma and carcinoma cell lines reacted similarly with the alpha-V group. The reaction with 17E6 is summarized (FIG. 3). The initial attachment to vitronectin of cells reacting in FACS with 17E6 was strongly blocked by that antibody, but only weakly affected by the control antibody 14E2 (FIG. 3). Other members of the alpha-V group were less potent (data not shown). The vigorous attachment of murine cell B16F10 on vitronectin was not affected by 17E6 and B16F10 did not react with 17E6 in FACS. As predicted (Cheresh and Harper, 1987), B16F10 attachment to vitronectin was sensitive to micromolar concentrations of RGD-peptides, suggesting the presence of functional surface .alpha.v.beta.3 (SLG and B. Diefenbach). Thus, 17E6 and the alpha.-V group reacted with human but not mouse .alpha.v.

Detailed Description Text (16):

The invention investigated the effect of the .alpha.v-blocking antibody 17E6 on the subcutaneous development of M21 tumors in BALB/c nu/nu mice (FIG. 7). In animal models, the development of M21 tumors in nude mice has been correlated with the cell surface expression of .alpha.v-series integrins (see Background). M21 cells were subcutaneously co-injected and endotoxin-free antibodies. 17E6 consistently (4/4 experiments) blocked the subcutaneous development of M21 tumors (FIG. 7a). No tumors (0/32) have taken in the presence of 17E6, and the animals still remain tumor free--now in excess of 6 months. Control tumor take was 75-90%. Non-blocking antibodies against the .alpha.v-chain itself and control antibodies against the melanoma cell surface showed variable and inconsistent effects on tumor development. In 14E2 treated controls, take of tumors was reduced depending on experiment 30-60%, but remaining tumors grew as the untreated controls and, like the controls, these animals had pulmonary micro-metastases revealed when the lungs were brought into tissue culture (not shown). By contrast, 17E6 treated animals had neither subcutaneous tumors nor metastases in lungs, liver, kidney, spleen, colon, stomach, nor in thoracic or abdominal body cavities when sacrificed at 6 months. The .alpha.v.beta.3-deficient line M21-L grew more slowly subcutaneously than M21, and was unaffected by 17E6. M21-L controls treated with 14E2 had a take reduced in comparison to untreated animals, similar to that seen in M21 cells (FIG. 7b).

Detailed Description Text (17):

The growth of M21 and M21-L and the effect of the antibody 17E6 were compared in an "experimental metastasis" tail-vein injection model. M21 formed many colonies in a dose dependent manner, while M21-L formed significantly fewer colonies, but did form lung nodules when injected at higher dosage (FIG. 7c). In other words, tumor growth in the lungs was also enhanced by the presence of cell surface .alpha.v-integrins, and pre-incubation of M21 with 17E6 reduced (by 90%) the numbers of tumor colonies that formed. Interestingly, the level of tumor formation was similar to those achieved by M21-L cells in the same experiment. The antibody did not altering the numbers of animals in which the tumor grew (Table 3).

Detailed Description Text (23):

Having tested the effects on cell proliferation, ADCC and AECM, it was examined whether the levels of DNA synthesis in M21 cells were affected by the MAbs. 17E6, 14E2, and LM609 at 0.5 M had no effect on thymidine incorporation (FIG. 9). DNA-synthesis in M21-L, M21-L4 and M21-L-IIb cells were also unaffected by the antibodies. M21-L and M21-L-IIb react neither with 17E6 nor LM609, but do react with 14E2 (FIG. 1). Many other melanoma cell lines were also tested and their DNA synthesis was shown not to be obviously affected by the alpha-V group (not shown).

Detailed Description Text (39):

Tumor progression and metastasis is classically a disease where cells escape normal growth and adhesion controls and invade, migrate, attach and grow at an inappropriate site. Integrins are now known to control many cell adhesion events, and adhesion can in turn regulate mechanistically interwoven events including growth, differentiation, cell movement and the activity of protease networks, developmental events which are reiterated in the metastatic cascade (Liotta et al.,

1991; Stetler Stevenson et al., 1993; Fidler, 1988). In this study antibodies are described directed against human .alpha.V-series integrins one of which, 17E6, perturbs initial cell attachment, disrupts stable .alpha.v-ligand interactions and interferes with human melanoma development in an in vivo animal model (Fidler, 1986). In biochemical analyses the alpha-V group antibodies showed reaction patterns closely related to LM142--a well defined antibody body to human .alpha.v--but distinct from the reaction patterns of .alpha.V.beta.3-specific (LM609), .alpha.V.beta.5-specific (P5H9) and from other defined anti-integrin antibodies. Thus, the alpha-V group antibodies are likely to recognize the human .alpha.V-integrin chain. cDNA cloning of the 17E6 immunoglobulin transcripts revealed unique, unambiguous sequences. The heavy chain variable sequences were characteristic of Kabat group IIb immunoglobulins and the light chain sequences characteristic of Kabat group V (Kabat et al., 1987). Thus, the antibody is uniquely defined.

Detailed Description Text (43):

Growth of M21 tumors in nude mice depends strongly on .alpha.V-integrins (Felding-Habermann et al., 1992; Sanders et al., 1992). As 17E6 could modulate stable .alpha.V-ligand interactions and had a long term effect its effect on tumor development was examined. It was found that 17E6 blocked the development of subcutaneous M21 tumors in nude mice, thus strongly supporting the studies of Felding-Habermann et al. In addition, it could be shown that .alpha.v also promoted, and 17E6 inhibited, the development of M21 as experimental lung metastases. This invention has thus independently confirmed the important earlier study, and extended it by using syngeneic antibody-mediated "therapy" with 17E6. These results emphasize the importance of .alpha.V-integrins in the development of the M21 tumor, and eliminate the possibility that the earlier results arose as selection artefacts of cloning.

Detailed Description Text (45):

It is interesting to compare 17E6 with RGD-peptides. In the B16F10-C57blk6 murine melanoma model coinjecting peptides inhibited the development of B16-F10 pulmonary tumors (Humphries et al., 1986; Hardan et al., 1993). With the same assumptions as for 17E6, .about.100 .mu.M RGD-peptide was present (Hardan et al., 1993), some two orders of magnitude over the dose required to block cell attachment to vitronectin. However, RGDS (SEQ ID NO:6) has a serum half-life of 8 min (Humphries et al., 1988). As a general therapeutic goal, it might be preferable to generate long-lived blockers for suppressing tumor development.

Detailed Description Text (127):

For experimental lung metastasis, cells were harvested (0.05% Trypsin/0.02% EDTA) and were injected into the tail vein of nude mice (0.5.times.10.sup.6 cells in 0.2 ml PBS++). 7 weeks later the animals were sacrificed, the lungs removed and fixed in a Bouins' solution, and the tumor foci on the surface of the lungs counted. For antibody treatment, harvested and washed cells were incubated with purified endotoxin free MAbs (70 .mu.g per 10.sup.6 cells 0.5 ml PBS++) for 30 min at 20.degree. C. in an end-over-end rotator before dilution to 0.5.times.10.sup.6 cells in 0.2 ml PBS and injection. Cells viability was assessed by Trypan blue dye exclusion before and after completing the injection schedule, where no significant difference was found (viability pre-injection=viability post injection .+-.5%). The tumor inhibition data was assessed using the 2-tailed Student T-test.

Detailed Description Paragraph Table (3):

TABLE 3 Inhibition of development of M21 tumor foci by 17E6 Mab in BALB/C nu/nu mice lung colonization "experimental metastasis" assay. Cells and Tumor Number of Tumor Foci & Treatment Take Mean .+-. SEM Median (Range) Control M21 (Control) 99
87 .+-. 110 30 (3-378) 700 M21 + 17E6 78 8 .+-. 7.7 5 (0.21) 9 M21-L 56 19 .+-. 22
8.5 (0-60) 22* Table 3: M21 and M21L cells were harvested with trypsin/EDTA, incubated with 17E6 antibody or control antibody, washed and injected into the tail vein of nude mice. 7 weeks later the animals were sacrificed and the lungs examined for surface tumor foci. Pretreatment with 17E6 lowered the numbers of foci that developed. Similar numbers of foci developed when M21L cells (which lack .alpha.v on the cell surface) were injected. * = Compared to control: not antibodydependent.

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Term	Documents
LUNG.DWPI,EPAB,JPAB,USPT.	46989
LUNGS.DWPI,EPAB,JPAB,USPT.	19933
PULMONARY.DWPI,EPAB,JPAB,USPT.	26668
PULMONARIES	0
PULMONARYS	0
CANCER.DWPI,EPAB,JPAB,USPT.	93005
CANCERS.DWPI,EPAB,JPAB,USPT.	21667
TUMOR.DWPI,EPAB,JPAB,USPT.	53934
TUMORS.DWPI,EPAB,JPAB,USPT.	33289
TUMOUR.DWPI,EPAB,JPAB,USPT.	22901
TUMOURS.DWPI,EPAB,JPAB,USPT.	12841
((LUNG OR PULMONARY) SAME (CANCER OR TUMOR OR TUMOUR) AND (BETA6)).USPT,JPAB,EPAB,DWPI.	2

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DATE: Saturday, October 12, 2002 [Printable Copy](#) [Create Case](#)

Set Name Query
side by side**Hit Count Set Name**
result set*DB=USPT,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ*L9 (lung or pulmonary) same (cancer or tumor or tumour) and (beta6) 2 L9L8 (lung or pulmonary) same (cancer or tumor or tumour) and (alphaV or 'alpha v') 47 L8L7 L6 and (alphaV or 'alpha v') 1 L7L6 L5 and (pulmonary or lung) same (fibrosis or fibrotic) 62 L6L5 rgd and (fibrosis or fibrotic) 174 L5*DB=USPT; PLUR=YES; OP=ADJ*L4 (alphav or 'alpha v') and (fibrosis or fibrotic) 16 L4*DB=USPT,EPAB,DWPI; PLUR=YES; OP=ADJ*L3 (alphav or 'alpha v') same (lung or pulmonary) 10 L3L2 (alphav or 'alpha v') same (antibod\$) and (treat\$ or therap\$ or suppress\$ or inhibit\$ or prevent\$) same (fibrosis or fibrotic) 2 L2L1 (alphav or 'alpha v') same (fibrosis or fibrotic) 14 L1

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12oct02 09:20:50 User208760 Session D2187.1
\$0.35 0.100 DialUnits File1
\$0.35 Estimated cost File1
\$0.35 Estimated cost this search
\$0.35 Estimated total session cost 0.100 DialUnits

File 410:Chronolog(R) 1981-2002/Sep
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? begin 5,73,155,399

12oct02 09:20:55 User208760 Session D2187.2
\$0.00 0.073 DialUnits File410
\$0.00 Estimated cost File410
\$0.01 TELNET
\$0.01 Estimated cost this search
\$0.36 Estimated total session cost 0.173 DialUnits

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*File 73: Alert feature enhanced for multiple files, duplicates removal, customized scheduling. See HELP ALERT.

File 155:MEDLINE(R) 1966-2002/Oct W1

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? s (beta6 or beta(w)6) and (fibrosis or fibrotic)

536 BETA6

1764082 BETA

4130995 6

3667 BETA(W)6

186165 FIBROSIS

16604 FIBROTIC

S1 43 (BETA6 OR BETA(W)6) AND (FIBROSIS OR FIBROTIC)

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...completed examining records

S2 26 RD S1 (unique items)

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2/3/1 (Item 1 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)
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13692381 BIOSIS NO.: 200200321202

Transforming growth factor beta (TGFbeta) dependent and independent pathways of induction of tubulointerstitial fibrosis in alphavbeta6 -/- mice.

Handwritten: 2V96
FIBROSIS

AUTHOR: Ma L-J(a); Donnert E; Sheppard D; Fogo A B
AUTHOR ADDRESS: (a)Dept. of Pathology, Vanderbilt University, Nashville, TN
**USA
JOURNAL: Journal of the American Society of Nephrology 12 (Program and
Abstract Issue):p819A September, 2001
MEDIUM: print
CONFERENCE/MEETING: ASN (American Society of Nephrology)/ISN (International
Society of Nephrology) World Congress of Nephrology San Francisco, CA, USA
October 10-17, 2001
ISSN: 1046-6673
RECORD TYPE: Citation
LANGUAGE: English

2/3/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2002 BIOSIS. All rts. reserv.

13546734 BIOSIS NO.: 200200175555
Spontaneous emphysema and upregulation of MMP-12 expression following
beta6 integrin inactivation.
AUTHOR: Morris David G(a); Kaminski Naftali; Huang Xiaozhu; Shapiro Steven
D; Sheppard Dean
AUTHOR ADDRESS: (a)Department of Medicine, Lung Biology Center, University
of California, San Francisco, San Francisco, CA, 94143**USA
JOURNAL: Molecular Biology of the Cell 11 (Supplement):p257a Dec., 2000
MEDIUM: print
CONFERENCE/MEETING: 40th American Society for Cell Biology Annual Meeting
San Francisco, CA, USA December 09-13, 2000
ISSN: 1059-1524
RECORD TYPE: Citation
LANGUAGE: English

2/3/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2002 BIOSIS. All rts. reserv.

13533818 BIOSIS NO.: 200200162639
Molecular and structural consequences of early renal allograft injury.
AUTHOR: Baboolal Keshwar(a); Jones Geraint A; Janezic Alenka; Griffiths
David R; Jurewicz Wieslaw A
AUTHOR ADDRESS: (a)Welsh Transplant Research Group, University Hospital of
Wales, Heath Park, Cardiff, CF14 4XW**UK E-Mail: Baboolalk@cf.ac.uk
JOURNAL: Kidney International 61 (2):p686-696 February, 2002
MEDIUM: print
ISSN: 0085-2538
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

2/3/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2002 BIOSIS. All rts. reserv.

13339516 BIOSIS NO.: 200100546665
Increased expression of tenascin-C-binding epithelial integrins in human
bullous keratopathy corneas.
AUTHOR: Ljubimov Alexander V(a); Saghizadeh Mehrnoosh; Pytela Robert;
Sheppard Dean; Kenney M Cristina
AUTHOR ADDRESS: (a)Ophthalmology Research Laboratories, Cedars-Sinai
Medical Center, 8700 Beverly Boulevard, Davis-5069, Los Angeles, CA,
90048: ljubimov@cshs.org**USA

JOURNAL: Journal of Histochemistry and Cytochemistry 49 (11):p1341-1350
November, 2001
MEDIUM: print
ISSN: 0022-1554
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

2/3/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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12921523 BIOSIS NO.: 200100128672
Protection against unilateral ureteral obstruction (UUO) induced
tubulointerstitial **fibrosis** in α - β mice.
AUTHOR: Ma L-J(a); Donnert E; Sheppard D; Fogo A B
AUTHOR ADDRESS: (a)Department of Pathology, Vanderbilt University,
Nashville, TN**USA
JOURNAL: Laboratory Investigation 81 (1):p189A January, 2001
MEDIUM: print
CONFERENCE/MEETING: Annual Meeting of the United States and Canadian
Academy of Pathology Atlanta, Georgia, USA March 03-09, 2001
ISSN: 0023-6837
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English

2/3/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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12661639 BIOSIS NO.: 200000415141
Mesenchymal regulation of alveolar repair in pulmonary **fibrosis**.
AUTHOR: Fang Kenneth C(a)
AUTHOR ADDRESS: (a)Cardiovascular Research Institute, University of
California, San Francisco, San Francisco, CA, 94143-0911**USA
JOURNAL: American Journal of Respiratory Cell and Molecular Biology 23 (2
) :p142-145 August, 2000
MEDIUM: print
ISSN: 1044-1549
DOCUMENT TYPE: Article
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English

2/3/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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12410227 BIOSIS NO.: 200000163729
Global analysis of gene expression in pulmonary **fibrosis** reveals
distinct programs regulating lung inflammation and **fibrosis**.
AUTHOR: Kaminski Naftali; Allard John D; Pittet Jean F; Zuo Fengrong;
Griffiths Mark JD; Morris David; Huang Xiaozhu; Sheppard Dean; Heller
Renu A(a)
AUTHOR ADDRESS: (a)Roche Bioscience, 3401 Hillview Avenue, Palo Alto, CA,
94304**USA
JOURNAL: Proceedings of the National Academy of Sciences of the United
States of America. 97 (4):p1778-1783 Feb. 15, 2000
ISSN: 0027-8424

DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

2/3/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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11896894 BIOSIS NO.: 199900143003
The integrin alphavbeta6 binds and activates latent TGFbeta1: A mechanism for regulating pulmonary inflammation and **fibrosis**.
AUTHOR: Munger John S; Huang Xiaozhu; Kawakatsu Hisaaki; Griffiths Mark J D ; Dalton Stephen L; Wu Jianfeng; Pittet Jean-Francois; Kaminski Naftali; Garat Chrystelle; Matthay Michael A; Rifkin Daniel B; Sheppard Dean(a)
AUTHOR ADDRESS: (a)Lung Biology Center, Department Medicine, University California San Francisco, San Francisco, CA**USA
JOURNAL: Cell 96 (3):p319-328 Feb. 5, 1999
ISSN: 0092-8674
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

2/3/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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11092846 BIOSIS NO.: 199799713991
Expression of integrin cell adhesion receptors during human airway epithelial repair in vivo.
AUTHOR: Pilewski Joseph M(a); Latoche Joseph D; Arcasoy Selim M; Albelda Steven M
AUTHOR ADDRESS: (a)Div. Pulmonary Med., Univ. Pittsburgh, 440 Scaife Hall, 3550 Terrace St., Pittsburgh, PA 15261**USA
JOURNAL: American Journal of Physiology 273 (1 PART 1):pL256-L263 1997
ISSN: 0002-9513
RECORD TYPE: Abstract
LANGUAGE: English

2/3/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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10773331 BIOSIS NO.: 199799394476
Inactivation of the **beta-6** integrin subunit gene protects against bleomycin-induced pulmonary **fibrosis**.
AUTHOR: Griffiths Mark; Huang Xiao-Zhu; Wu Jian Geng; Sheppard Dean
AUTHOR ADDRESS: Lung Biol. Cent., Univ. Calif., San Francisco, CA 94143** USA
JOURNAL: Molecular Biology of the Cell 7 (SUPPL.):p166A 1996
CONFERENCE/MEETING: Annual Meeting of the 6th International Congress on Cell Biology and the 36th American Society for Cell Biology San Francisco, California, USA December 7-11, 1996
ISSN: 1059-1524
RECORD TYPE: Citation
LANGUAGE: English

2/3/11 (Item 11 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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10714773 BIOSIS NO.: 199799335918
Induction of interstitial pneumonia in autoimmune mice by intratracheal
administration of superantigen staphylococcal enterotoxin B.
AUTHOR: Shinbori Toshifumi; Matsuki Misae; Suga Moritaka; Kakimoto Kiichi;
Ando Masayuki(a)
AUTHOR ADDRESS: (a)First Dep. Internal Med., Kumamoto Univ. Sch. Med.,
1-1-1 Honjo, Kumamoto 860**Japan
JOURNAL: Cellular Immunology 174 (2):p129-137 1996
ISSN: 0008-8749
RECORD TYPE: Abstract
LANGUAGE: English

2/3/12 (Item 12 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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10431140 BIOSIS NO.: 199699052285
Cytokine gene expression in cirrhotic and non-cirrhotic human liver.
AUTHOR: Llorente Luis(a); Richaud-Patin Yvonne; Alcocer-Castillejos Natasha
; Ruiz-Soto Rodrigo; Mercado Miguel Angel; Orozco Hector;
Gamboa-Dominguez Armando; Alcocer-Varela Jorge
AUTHOR ADDRESS: (a)Dep. Immunology Rheumatology, Instituto Nacional de la
Nutricion Salvador Zubiran, Vasco de Quir**Mexico
JOURNAL: Journal of Hepatology 24 (5):p555-563 1996
ISSN: 0168-8278
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

2/3/13 (Item 13 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2002 BIOSIS. All rts. reserv.

10293223 BIOSIS NO.: 199698748141
Interleukin 1 and tumor necrosis factors in obese alcoholics compared with
normal-weight patients.
AUTHOR: Bunout Daniel(a); Munoz Carlos; Lopez Marcelo; Pia De La Maza Maria
; Schlesinger Liana; Hirsch Sandra; Pettermann Margarita
AUTHOR ADDRESS: (a)INTA, Univ. Chile, P.O. Box 138-11, Santiago**Chile
JOURNAL: American Journal of Clinical Nutrition 63 (3):p373-376 1996
ISSN: 0002-9165
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

2/3/14 (Item 14 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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09114307 BIOSIS NO.: 199497122677
Rapid detection of single nucleotide deletions: Application to the
beta 6 (-A) mutation of the beta-globin gene and to cystic
fibrosis.
AUTHOR: Romey Marie-Catherine; Aguilar-Martinez Patricia; Demaille Jacques;
Claustres Mireille(a)
AUTHOR ADDRESS: (a)INSERM U249/CNRS UPR 9008, Laboratoire de Biochimie
Genetique, Institut de Biologie, Boulevard H**France
JOURNAL: Human Genetics 92 (6):p627-628 1993
ISSN: 0340-6717
DOCUMENT TYPE: Article

RECORD TYPE: Abstract
LANGUAGE: English

2/3/15 (Item 15 from file: 5)
DIALOG(R)File 5:BIOSIS Previews(R)
(c) 2002 BIOSIS. All rts. reserv.

08802020 BIOSIS NO.: 199395091371
Quantitation and structures of oligosaccharide chains in human trachea
mucin glycoproteins.
AUTHOR: Sangadala Sreedhara; Bhat U Ramadas; Mendicino Joseph(a)
AUTHOR ADDRESS: (a)Dep. Biochem., University Georgia, Athens, Georgia 30602
JOURNAL: Molecular and Cellular Biochemistry 118 (1):p75-90 1992
ISSN: 0300-8177
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

2/3/16 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2002 Elsevier Science B.V. All rts. reserv.

11254342 EMBASE No: 2001269685
Integrin-mediated activation of transforming growth factor-betaSUP1 in
pulmonary **fibrosis**
Sheppard D.
Dr. D. Sheppard, Lung Biology Center, UCSF Box 0854, San Francisco, CA
94143 United States
Chest (CHEST) (United States) 2001, 120/SUPPL. (49S-53S)
CODEN: CHETB ISSN: 0012-3692
DOCUMENT TYPE: Journal ; Conference Paper
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 28

2/3/17 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12993001 21843125 PMID: 11854220
Pulmonary inflammation induced by Pseudomonas aeruginosa
lipopolysaccharide, phospholipase C, and exotoxin A: role of interferon
regulatory factor 1.
Wieland Catharina W; Siegmund Britta; Senaldi Giorgio; Vasil Michael L;
Dinarello Charles A; Fantuzzi Giamila
Department of Medicine, University of Colorado Health Sciences Center,
Denver, Colorado 80262, USA.
Infection and immunity (United States) Mar 2002, 70 (3) p1352-8,
ISSN 0019-9567 Journal Code: 0246127
Contract/Grant No.: AI-15614; AI; NIAID; HL62608; HL; NHLBI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

2/3/18 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11302659 21344899 PMID: 11451914
Integrin-mediated activation of transforming growth factor-beta(1) in
pulmonary **fibrosis**.
Sheppard D

Lung Biology Center, Center for Occupational and Environmental Health,
Cardiovascular Research Institute, Department of Medicine, University of
California, San Francisco, San Francisco, CA 94143, USA.

Chest (United States) Jul 2001, 120 (1 Suppl) p49S-53S, ISSN
0012-3692 Journal Code: 0231335
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

2/3/19 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10155488 99148265 PMID: 10025398

The integrin alpha v **beta 6** binds and activates latent TGF
beta 1: a mechanism for regulating pulmonary inflammation and
fibrosis.

Munger J S; Huang X; Kawakatsu H; Griffiths M J; Dalton S L; Wu J; Pittet
J F; Kaminski N; Garat C; Matthay M A; Rifkin D B; Sheppard D

Department of Medicine, and Kaplan Cancer Center, New York University
School of Medicine, New York 10016-6402, USA.

Cell (UNITED STATES) Feb 5 1999, 96 (3) p319-28, ISSN 0092-8674
Journal Code: 0413066

Contract/Grant No.: HL47412; HL; NHLBI; HL53949; HL; NHLBI; HL56385; HL;
NHLBI; +

Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

2/3/20 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

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136084699 CA: 136(6)84699x PATENT

Treatment of acute lung injury and fibrosis with antagonists of avbeta6

INVENTOR(AUTHOR): Huang, Xiaozhu; Sheppard, Dean; Pytela, Robert

LOCATION: USA

PATENT: U.S. Pat. Appl. Publ. ; US 20020004482 A1 DATE: 20020110

APPLICATION: US 130870 (19980807) *US PV55060 (19970808)

PAGES: 9 pp., Cont.-in-part of U.S. Provisional 55,060. CODEN: USXXCO

LANGUAGE: English CLASS: 514012000; A61K-038/16A; A61K-031/70B;

C07K-017/00B; C07K-014/00B

2/3/21 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

(c) 2002 American Chemical Society. All rts. reserv.

136015216 CA: 136(2)15216a PATENT

Methods for identifying modulators of the interaction between LAP
(latency associated peptide) and integrin .alpha.v.beta.3, and medical use
thereof

INVENTOR(AUTHOR): Ludbrook, Steven; Barry, Simon; Horgan, Carmel; Miller,
David

LOCATION: UK,

ASSIGNEE: Glaxo Group Limited

PATENT: PCT International ; WO 200190186 A1 DATE: 20011129

APPLICATION: WO 2001GB2352 (20010525) *GB 200012991 (20000526) *GB
2001286 (20010105)

PAGES: 44 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C07K-014/705A;
C07K-014/475B; G01N-033/68B; G01N-033/566B; A61K-035/00B; A61K-038/00B

DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; US; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; MZ; SD; SL; SZ; TZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG

2/3/22 (Item 3 from file: 399)

DIALOG(R)File 399:CA SEARCH(R)

(c) 2002 American Chemical Society. All rts. reserv.

134160854 CA: 134(12)160854v JOURNAL

Transgenic and knockout mouse models of pulmonary inflammatory diseases

AUTHOR(S): Griffiths, Mark

LOCATION: Adult Intensive Care Unit, Royal Brompton Hospital, London, UK, SW3 6NP

JOURNAL: Biomed. Health Res. DATE: 2000 VOLUME: 34 NUMBER: Acute Lung Injury: From Inflammation to Repair PAGES: 93-104 CODEN: BIHREN ISSN: 0929-6743 LANGUAGE: English PUBLISHER: IOS Press

2/3/23 (Item 4 from file: 399)

DIALOG(R)File 399:CA SEARCH(R)

(c) 2002 American Chemical Society. All rts. reserv.

134086549 CA: 134(7)86549f PATENT

Preparation of cyclic peptides for use as inhibitors of integrin

.alpha.v.beta.6

INVENTOR(AUTHOR): Jonczyk, Alfred; Diefenbach, Beate; Goodman, Simon

LOCATION: Germany,

ASSIGNEE: Merck Patent G.m.b.H.

PATENT: Germany Offen. ; DE 19933173 A1 DATE: 20010118

APPLICATION: DE 19933173 (19990715)

PAGES: 20 pp. CODEN: GWXXBX LANGUAGE: German CLASS: C07K-007/64A; A61K-038/12B

2/3/24 (Item 5 from file: 399)

DIALOG(R)File 399:CA SEARCH(R)

(c) 2002 American Chemical Society. All rts. reserv.

134042449 CA: 134(4)42449u PATENT

Synthesis of peptide inhibitors of integrin .alpha.v.beta.6

INVENTOR(AUTHOR): Jonczyk, Alfred; Diefenbach, Beate; Groth, Ulrich; Zischinsky, Gunther

LOCATION: Germany,

ASSIGNEE: Merck Patent G.m.b.H.

PATENT: Germany Offen. ; DE 19929410 A1 DATE: 20001228

APPLICATION: DE 19929410 (19990626)

PAGES: 34 pp. CODEN: GWXXBX LANGUAGE: German CLASS: C07K-007/06A; A61K-038/08B

2/3/25 (Item 6 from file: 399)

DIALOG(R)File 399:CA SEARCH(R)

(c) 2002 American Chemical Society. All rts. reserv.

133068988 CA: 133(6)68988y PATENT

Integrin .alpha.v.beta.6 inhibitor peptides, and therapeutic use

INVENTOR(AUTHOR): Diefenbach, Beate; Jonczyk, Alfred; Kraft, Sabine; Mehta, Ray

LOCATION: Germany,
ASSIGNEE: Merck Patent G.m.b.H.
PATENT: PCT International ; WO 200037487 A1 DATE: 20000629
APPLICATION: WO 99EP9842 (19991211) *DE 19858587 (19981219)
PAGES: 37 pp. CODEN: PIXXD2 LANGUAGE: German CLASS: C07K-007/06A;
C07K-007/08B; C12N-015/10B; A61K-038/04B; A61P-007/02B
DESIGNATED COUNTRIES: AE; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH;
CN; CU; CZ; DE; DK; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS;
JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MD; MG; MK; MN; MW; MX;
NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; UA; UG; US;
UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM
DESIGNATED REGIONAL: GH; GM; KE; LS; MW; SD; SL; SZ; TZ; UG; ZW; AT; BE;
CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF;
CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG

2/3/26 (Item 7 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2002 American Chemical Society. All rts. reserv.

130195758 CA: 130(15)195758m PATENT
Treatment of acute lung injury and fibrosis with antagonists of
.alpha.v.beta.6
INVENTOR(AUTHOR): Huang, Xiaozhu; Sheppard, Dean; Pytela, Robert
LOCATION: USA
ASSIGNEE: The Regents of the University of California
PATENT: PCT International ; WO 9907405 A1 DATE: 19990218
APPLICATION: WO 98US16439 (19980807) *US 55060 (19970808)
PAGES: 23 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-038/16A;
A61K-038/17B; A61K-039/395B; A61K-048/00B; C07H-021/04B; C07K-014/435B;
C07K-014/47B; C07K-016/18B; C07K-016/28B DESIGNATED COUNTRIES: AL; AM; AT;
AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CU; CZ; DE; DK; EE; ES; FI; GB; GE;
GH; GM; HR; HU; ID; IL; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU;
LV; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL;
TJ; TM; TR; TT; UA; UG; UZ; VN; YU; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM
DESIGNATED REGIONAL: GH; GM; KE; LS; MW; SD; SZ; UG; ZW; AT; BE; CH; CY;
DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI;
CM; GA; GN; GW; ML; MR; NE; SN; TD; TG
?
? t s2/7/23,24,25

2/7/23 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2002 American Chemical Society. All rts. reserv.

134086549 CA: 134(7)86549f PATENT
Preparation of cyclic peptides for use as inhibitors of integrin
.alpha.v.beta.6
INVENTOR(AUTHOR): Jonczyk, Alfred; Diefenbach, Beate; Goodman, Simon
LOCATION: Germany,
ASSIGNEE: Merck Patent G.m.b.H.
PATENT: Germany Offen. ; DE 19933173 A1 DATE: 20010118
APPLICATION: DE 19933173 (19990715)
PAGES: 20 pp. CODEN: GWXXBX LANGUAGE: German CLASS: C07K-007/64A;
A61K-038/12B
SECTION:
CA234003 Amino Acids, Peptides, and Proteins
CA201XXX Pharmacology
CA263XXX Pharmaceuticals
IDENTIFIERS: integrin alphav beta6 inhibitor peptide prepn therapeutic
DESCRIPTORS:
Artery,disease...
coronary; prepn. of cyclic peptides for use as inhibitors of integrin
.alpha.v.beta.6 in treatment of

Peptides, preparation...

cyclic; prepn. of cyclic peptides for use as inhibitors of integrin .alpha.v.beta.6 in treatment of disease

Heart, disease...

infarction; prepn. of cyclic peptides for use as inhibitors of integrin .alpha.v.beta.6 in treatment of

Arteriosclerosis... Fibrosis... Infection... Inflammation... Neoplasm...

Osteoporosis... Psoriasis... Thrombosis... Wound healing...

prepn. of cyclic peptides for use as inhibitors of integrin .alpha.v.beta.6 in treatment of

Integrins... Receptors...

prepn. of cyclic peptides for use as inhibitors of integrin .alpha.v.beta.6 in treatment of disease

CAS REGISTRY NUMBERS:

116821-47-7 317366-48-6P 317366-49-7P 317366-50-0P 317366-51-1P
317366-52-2P 317366-53-3P 317366-54-4P 317366-55-5P 317366-56-6P
317366-57-7P 317366-58-8P 317366-59-9P 317366-60-2P 317366-61-3P
317366-62-4P 317366-63-5P 317366-64-6P 317366-65-7P 317366-66-8P
317366-67-9P 317366-68-0P 317366-69-1P 317366-70-4P 317366-71-5P
317366-72-6P 317366-73-7P 317366-74-8P 317366-75-9P 317366-76-0P
317366-77-1P 317366-78-2P 317366-79-3P 317366-80-6P prepn. of
cyclic peptides for use as inhibitors of integrin .alpha.v.beta.6 in
treatment of
116821-47-7DP resin-bound, prepn. of cyclic peptides for use as inhibitors
of integrin .alpha.v.beta.6 in treatment of

2/7/24 (Item 5 from file: 399)

DIALOG(R) File 399:CA SEARCH(R)

(c) 2002 American Chemical Society. All rts. reserv.

134042449 CA: 134(4)42449u PATENT

Synthesis of peptide inhibitors of integrin .alpha.v.beta.6

INVENTOR(AUTHOR): Jonczyk, Alfred; Diefenbach, Beate; Groth, Ulrich;
Zischinsky, Gunther

LOCATION: Germany,

ASSIGNEE: Merck Patent G.m.b.H.

PATENT: Germany Offen. ; DE 19929410 A1 DATE: 20001228

APPLICATION: DE 19929410 (19990626)

PAGES: 34 pp. CODEN: GWXXBX LANGUAGE: German CLASS: C07K-007/06A;
A61K-038/08B

SECTION:

CA234003 Amino Acids, Peptides, and Proteins

CA201XXX Pharmacology

CA263XXX Pharmaceuticals

IDENTIFIERS: integrin alphav beta6 inhibitor peptide prepn therapeutic
solid phase

DESCRIPTORS:

Artery, disease...

coronary; prepn. of peptide inhibitors of integrin .alpha.v.beta.6 for
treatment of disease

Heart, disease...

infarction; prepn. of peptide inhibitors of integrin .alpha.v.beta.6
for treatment of disease

Solid phase synthesis...

peptide; prepn. of peptide inhibitors of integrin .alpha.v.beta.6 for
treatment of disease

Arteriosclerosis... Drug delivery systems... Drugs... Fibrosis... Infection
... Inflammation... Integrins... Neoplasm... Osteoporosis...

Peptides, preparation... Psoriasis... Receptors... Thrombosis... Wound
healing...

prepn. of peptide inhibitors of integrin .alpha.v.beta.6 for treatment
of disease

CAS REGISTRY NUMBERS:

278777-44-9P 313245-97-5P 313246-01-4P 313246-05-8P 313246-09-2P
 313246-13-8P 313246-17-2P 313246-21-8P 313246-25-2P 313246-28-5P
 313246-31-0P 313246-34-3P 313246-39-8P 313246-41-2P 313246-44-5P
 313246-47-8P 313246-50-3P 313246-53-6P 313246-58-1P 313246-61-6P
 313246-64-9P 313246-67-2P 313246-70-7P 313246-75-2P 313246-78-5P
 313246-81-0P 313246-84-3P 313246-87-6P 313246-90-1P 313246-93-4P
 313246-96-7P 313246-99-0P 313247-02-8P 313247-05-1P 313247-09-5P
 313247-13-1P 313247-17-5P 313247-21-1P 313247-24-4P 313247-28-8P
 313247-31-3P 313247-33-5P 313247-36-8P 313247-39-1P 313247-42-6P
 313247-45-9P 313247-47-1P 313247-50-6P 313247-53-9P 313247-56-2P
 313247-59-5P 313247-62-0P 313247-65-3P 313247-68-6P 313247-72-2P
 313247-75-5P 313247-78-8P 313247-83-5P 313247-86-8P 313247-89-1P
 313247-91-5P 313247-93-7P 313247-95-9P prepn. of peptide inhibitors
 of integrin .alpha.v.beta.6 for treatment of disease
 313245-92-0DP resin-bound, prepn. of peptide inhibitors of integrin
 .alpha.v.beta.6 for treatment of disease

2/7/25 (Item 6 from file: 399)
 DIALOG(R)File 399:CA SEARCH(R)
 (c) 2002 American Chemical Society. All rts. reserv.

133068988 CA: 133(6)68988y PATENT
 Integrin .alpha.v.beta.6 inhibitor peptides, and therapeutic use
 INVENTOR(AUTHOR): Diefenbach, Beate; Jonczyk, Alfred; Kraft, Sabine;
 Mehta, Ray
 LOCATION: Germany,
 ASSIGNEE: Merck Patent G.m.b.H.
 PATENT: PCT International ; WO 200037487 A1 DATE: 20000629
 APPLICATION: WO 99EP9842 (19991211) *DE 19858587 (19981219)
 PAGES: 37 pp. CODEN: PIXXD2 LANGUAGE: German CLASS: C07K-007/06A;
 C07K-007/08B; C12N-015/10B; A61K-038/04B; A61P-007/02B
 DESIGNATED COUNTRIES: AE; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH;
 CN; CU; CZ; DE; DK; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS;
 JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MD; MG; MK; MN; MW; MX;
 NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; UA; UG; US;
 UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM
 DESIGNATED REGIONAL: GH; GM; KE; LS; MW; SD; SL; SZ; TZ; UG; ZW; AT; BE;
 CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF;
 CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG
 SECTION:
 CA201012 Pharmacology
 CA263XXX Pharmaceuticals
 IDENTIFIERS: integrin alphav beta6 inhibitor peptide therapeutic
 DESCRIPTORS:
 Integrins...
 .alpha.v.beta.6; integrin .alpha.v.beta.6 inhibitor peptides, and
 therapeutic use
 Drug delivery systems...
 capsules; integrin .alpha.v.beta.6 inhibitor peptides, and therapeutic
 use
 Artery,disease...
 coronary; integrin .alpha.v.beta.6 inhibitor peptides, and therapeutic
 use
 Drug delivery systems...
 dragees; integrin .alpha.v.beta.6 inhibitor peptides, and therapeutic
 use
 Heart,disease...
 infarction; integrin .alpha.v.beta.6 inhibitor peptides, and
 therapeutic use
 Drug delivery systems...
 inhalants; integrin .alpha.v.beta.6 inhibitor peptides, and therapeutic
 use
 Drug delivery systems...

injections; integrin .alpha.v.beta.6 inhibitor peptides, and
 therapeutic use
 Antiarteriosclerotics... Anticoagulants... Antitumor agents...
 Anti-infective agents... Anti-inflammatory agents... Cardiovascular agents
 ... DNA... Drug delivery systems... Envelope proteins... Fibrosis... Gene
 therapy... Peptides,biological studies... Psoriasis... Viral DNA... Wound
 healing promoters...
 integrin .alpha.v.beta.6 inhibitor peptides, and therapeutic use
 Drug delivery systems...
 liposomes; integrin .alpha.v.beta.6 inhibitor peptides, and therapeutic
 use
 Drug delivery systems...
 ointments; integrin .alpha.v.beta.6 inhibitor peptides, and therapeutic
 use
 Drug delivery systems...
 solns.; integrin .alpha.v.beta.6 inhibitor peptides, and therapeutic
 use
 Drug delivery systems...
 sprays; integrin .alpha.v.beta.6 inhibitor peptides, and therapeutic
 use
 Drug delivery systems...
 suppositories; integrin .alpha.v.beta.6 inhibitor peptides, and
 therapeutic use
 Drug delivery systems...
 tablets; integrin .alpha.v.beta.6 inhibitor peptides, and therapeutic
 use
 Osteoporosis...
 therapeutic agents; integrin .alpha.v.beta.6 inhibitor peptides, and
 therapeutic use
 CAS REGISTRY NUMBERS:
 222557-93-9P 268230-24-6P 268230-25-7P 268230-26-8P 278777-32-5P
 278777-33-6P 278777-34-7P 278777-35-8P 278777-36-9P 278777-37-0P
 278777-38-1P 278777-39-2P 278777-40-5P 278777-41-6P 278777-42-7P
 278777-43-8P 278777-44-9P 278777-45-0P 278777-46-1P integrin
 .alpha.v.beta.6 inhibitor peptides, and therapeutic use
 ?

Set	Items	Description
S1	43	(BETA6 OR BETA(W)6) AND (FIBROSIS OR FIBROTIC)
S2	26	RD S1 (unique items)

? s (beta6 or beta(w)6) and (lung or pulmonary) (20n)(cancer? or tumor? or tumour?)

Processing

	536	BETA6
	1764082	BETA
	4130995	6
	3667	BETA(W)6
	1013660	LUNG
	770351	PULMONARY
	1910461	CANCER?
	1945134	TUMOR?
	259811	TUMOUR?
	221642	(LUNG OR PULMONARY) (20N) ((CANCER? OR TUMOR?) OR TUMOUR?)
S3	16	(BETA6 OR BETA(W)6) AND (LUNG OR PULMONARY) (20N) (CANCER? OR TUMOR? OR TUMOUR?)

? rd s3

...completed examining records

S4	11	RD S3 (unique items)
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? t s4/3/all

4/3/1 (Item 1 from file: 5)
 DIALOG(R)File 5:Biosis Previews(R)
 (c) 2002 BIOSIS. All rts. reserv.

12862074 BIOSIS NO.: 200100069223

A role for the integrin alphavbeta8 in the negative regulation of epithelial cell growth.

AUTHOR: Cambier Stephanie; Mu De-zhi; O'Connell David; Boylen Kevin; Travis William; Liu Wei-hong; Broaddus V Courtney; Nishimura Stephen L(a)

AUTHOR ADDRESS: (a)Lung Biology Center, University of California at San Francisco, San Francisco, CA, 94143: cdog@itsa.ucsf.edu**USA

JOURNAL: Cancer Research 60 (24):p7084-7093 December 15, 2000

MEDIUM: print

ISSN: 0008-5472

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

SUMMARY LANGUAGE: English

4/3/2 (Item 2 from file: 5)
 DIALOG(R)File 5:Biosis Previews(R)
 (c) 2002 BIOSIS. All rts. reserv.

10426243 BIOSIS NO.: 199699047388

Neutrophil and cytokine activation with neonatal extracorporeal membrane oxygenation.

AUTHOR: Fortenberry James D(a); Bhardwaj Vijay; Niemer Paula; Cornish J Devn; Wright Jean A; Bland Lee

AUTHOR ADDRESS: (a)Dep. Pediatrics, Emory Univ. Sch. Med., 1405 Clifton Rd. NE, Atlanta, GA 30322**USA

JOURNAL: Journal of Pediatrics 128 (5 PART 1):p670-678 1996

ISSN: 0022-3476

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

4/3/3 (Item 3 from file: 5)
 DIALOG(R)File 5:Biosis Previews(R)
 (c) 2002 BIOSIS. All rts. reserv.

09917682 BIOSIS NO.: 199598372600

Expression of the **beta-6** integrin subunit in development,
neoplasia and tissue repair suggests a role in epithelial remodeling.

AUTHOR: Breuss J M; Gallo J; Delisser H M; Klimanskaya I V; Folkesson H G;
Pittet J F; Nishimura S L; Aldape K; Landers D V; Carpenter W; Gillett N;
Sheppard D; Matthay M A; Albelda S M; Kramer R H; Pytela R(a)

AUTHOR ADDRESS: (a)Lung Biol. Cent., Dep. Med., Univ. Calif. San Francisco,
San Francisco, CA 94143**USA

JOURNAL: Journal of Cell Science 108 (6):p2241-2251 1995

ISSN: 0021-9533

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

4/3/4 (Item 4 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

(c) 2002 BIOSIS. All rts. reserv.

09866238 BIOSIS NO.: 199598321156

Distribution of integrins alpha-v-**beta-6** and alpha-9-beta-1 and
their known ligands, fibronectin and tenascin, in human airways.

AUTHOR: Weinacker Ann; Ferrando Ronald; Elliott Mark; Hogg James; Balmes
John; Sheppard Dean(a)

AUTHOR ADDRESS: (a)Lung Biol. Cent., UCSF Box 0854, San Francisco, CA 94143
**USA

JOURNAL: American Journal of Respiratory Cell and Molecular Biology 12 (5)
):p547-557 1995

ISSN: 1044-1549

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

4/3/5 (Item 5 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

(c) 2002 BIOSIS. All rts. reserv.

08762949 BIOSIS NO.: 199395052300

T-cell subset analysis of Lewis lung carcinoma **tumor** rejection:

Heterogeneity of effectors and evidence for negative regulatory
lymphocytes correlating with metastasis.

AUTHOR: Gelber Gohava(a); Eisenbach Lea; Feldman Michael; Goodenow Robert S

AUTHOR ADDRESS: (a)Div. Immunol. Rheumatol., Stanford Univ. Sch. Med.,
Stanford, Calif. 94305

JOURNAL: Cancer Research 52 (23):p6507-6515 1992

ISSN: 0008-5472

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

4/3/6 (Item 6 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

(c) 2002 BIOSIS. All rts. reserv.

04629251 BIOSIS NO.: 000079042288

STUDIES ON SYNTHESIS AND ANTICANCER ACTIVITY OF SELECTED N-2

FLUOROETHYL-N-NITROSOUREAS

AUTHOR: JOHNSTON T P; KUSSNER C L; CARTER R L; FRYE J L; LOMAX N R; PLOWMAN
J; NARAYANAN V L

AUTHOR ADDRESS: DEV. THERAPEUTICS PROGRAM, DIV. CANCER TREATMENT, NATL.
CANCER INST., BETHESDA, MD. 20205.

JOURNAL: J MED CHEM 27 (11). 1984. 1422-1426. 1984
FULL JOURNAL NAME: Journal of Medicinal Chemistry
CODEN: JMCMA
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

4/3/7 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2002 Elsevier Science B.V. All rts. reserv.

10980880 EMBASE No: 2001028793
A role for the integrin alphavbeta38 in the negative regulation of
epithelial cell growth
Cambier S.; Mu D.-Z.; O'Connell D.; Boylen K.; Travis W.; Liu W.-H.;
Courtney Broadus C.; Nishimura S.L.
S.L. Nishimura, Lung Biology Center, Univ. of California at San
Francisco, Box 0854, San Francisco, CA 94143 United States
AUTHOR EMAIL: cdog@itsa.ucsf.edu
Cancer Research (CANCER RES.) (United States) 15 DEC 2000, 60/24
(7084-7093)
CODEN: CNREA ISSN: 0008-5472
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 73

4/3/8 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12993001 21843125 PMID: 11854220
Pulmonary inflammation induced by Pseudomonas aeruginosa
lipopolysaccharide, phospholipase C, and exotoxin A: role of interferon
regulatory factor 1.
Wieland Catharina W; Siegmund Britta; Senaldi Giorgio; Vasil Michael L;
Dinarello Charles A; Fantuzzi Giamila
Department of Medicine, University of Colorado Health Sciences Center,
Denver, Colorado 80262, USA.
Infection and immunity (United States) Mar 2002, 70 (3) p1352-8,
ISSN 0019-9567 Journal Code: 0246127
Contract/Grant No.: AI-15614; AI; NIAID; HL62608; HL; NHLBI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

4/3/9 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10155488 99148265 PMID: 10025398
The integrin alpha v beta 6 binds and activates latent TGF
beta 1: a mechanism for regulating pulmonary inflammation and fibrosis.
Munger J S; Huang X; Kawakatsu H; Griffiths M J; Dalton S L; Wu J; Pittet
J F; Kaminski N; Garat C; Matthay M A; Rifkin D B; Sheppard D
Department of Medicine, and Kaplan Cancer Center, New York University
School of Medicine, New York 10016-6402, USA.
Cell (UNITED STATES) Feb 5 1999, 96 (3) p319-28, ISSN 0092-8674
Journal Code: 0413066
Contract/Grant No.: HL47412; HL; NHLBI; HL53949; HL; NHLBI; HL56385; HL;
NHLBI; +
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM

Record type: Completed

4/3/10 (Item 3 from file: 155)
DIALOG(R) File 155:MEDLINE(R)

07519996 93046169 PMID: 1423297

T-cell subset analysis of Lewis lung carcinoma tumor rejection: heterogeneity of effectors and evidence for negative regulatory lymphocytes correlating with metastasis.

Gelber C; Eisenbach L; Feldman M; Goodenow R S

Division of Immunology and Rheumatology, Stanford University School of Medicine, California 94305.

Cancer research (UNITED STATES) Dec 1 1992, 52 (23) p6507-15, ISSN 0008-5472 Journal Code: 2984705R

Document type: Journal Article

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Main Citation Owner: NLM

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Integrin-binding domain of mitogen-activated protein kinases and its use for modulating cellular activity in cancer and other cells

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